# A Review on Advances in Multi-Target-Directed Ligands for Alzheimer's Disease

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# A Review on Advances in Multi-Target-Directed Ligands for Alzheimer's Disease

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**Abstract:**

*The multifactorial pathogenic causes of Alzheimer's disease (AD), a progressive neurodegenerative illness marked by cognitive decline and memory impairment, include tau hyperphosphorylation, beta-amyloid accumulation, oxidative stress, neuroinflammation, and cholinergic deficiency. Current therapies do not stop the course of the disease; they simply relieve its symptoms. Recent developments in the synthesis and design of multi-target-directed ligands (MTDLs), which seek to concurrently alter several AD-related pathways, are highlighted in this review. The focus is on hybrid compounds that combine anti-amyloid properties, metal chelation, antioxidant action, and acetylcholinesterase inhibition. Promising pharmacological profiles and neuroprotective potential are demonstrated by pyrimidine derivatives and coumarin-lipoic acid conjugates produced via nanoparticle catalysis. Furthermore, kinase inhibitors that target DYRK1A and associated enzymes provide fresh opportunities to modify tau pathogenesis. The development of successful disease-modifying treatments for AD is made possible by the combination of innovative synthetic processes, logical drug design, and thorough biological assessment. There is potential for bettering treatment results and tackling the intricate etiology of Alzheimer's disease with further study in this area.*

**Keywords:** Alzheimer's Disease Synthetic Chemistry; monoamine oxidase; acetylcholinesterase

## 1. INTRODUCTION

The most prevalent cause of dementia globally is Alzheimer's disease (AD), a crippling and progressive neurological illness. Memory loss, cognitive impairment, disorientation, linguistic abnormalities, and eventually loss of independence are its clinical hallmarks [1]. Two primary histopathological characteristics of AD are the intracellular buildup of neurofibrillary tangles made of hyperphosphorylated tau protein and the extracellular deposition of amyloid-beta (Aβ) plaques [2]. Apart from these characteristics, the pathophysiology of the illness is also influenced by oxidative stress, chronic neuroinflammation, mitochondrial dysfunction, and cholinergic deficiencies [3]. As the average lifespan rises, the worldwide burden of AD keeps growing. More than 55 million people worldwide already suffer from dementia, and by 2050, that figure is predicted to rise to over 150 million, putting a significant societal and financial strain on healthcare systems throughout the globe [4, 5].

Regretfully, there is still no treatment for AD despite a great deal of study. Current FDA-approved medications, such as NMDA receptor antagonist memantine and acetylcholinesterase inhibitors (donepezil, rivastigmine, and galantamine), merely relieve symptoms and do not slow the course of the illness [6]. These limitations have intensified the search for novel therapeutic agents that can modify the course of the disease. One of the most active areas in AD drug discovery is the development of synthetic compounds designed to target various pathological mechanisms simultaneously. The multi-target-directed ligand (MTDL) approach has gained traction, particularly because AD is a multifactorial disease involving interconnected biological pathways [7]. In this context, recent research has focused on designing hybrid molecules that can simultaneously inhibit acetylcholinesterase (AChE), prevent Aβ aggregation, scavenge free radicals, and modulate tau phosphorylation [8].

In recent years, chromones, coumarins, indoles, quinolines, flavonoids, and pyridine derivatives have been investigated as possible synthetic scaffolds [9]. These substances are frequently created using logical design techniques that include pharmacophores that target AChE in addition to other components that change illness. In an effort to stop metal-induced Aβ peptide aggregation, several hybrids additionally contain metal-chelating groups [10]. Numerous hybrid compounds have shown dual or triple actions, such as the simultaneous inhibition of monoamine oxidase (MAO-B) and AChE, which can improve cognition and protect against neurodegeneration [11, 12]. To address the behavioral and psychiatric problems frequently seen in AD patients, some compounds have also been created to interact with serotonin, dopamine, or histamine receptors [12].

This review aims to provide a comprehensive overview of the most recent advancements in the synthesis of compounds with potential therapeutic effects against Alzheimer's disease. Cholinesterase inhibition, antioxidant capacity, anti-amyloid aggregation, metal chelation, and neuroprotective qualities are just a few of the chemical families and scaffolds that have been investigated for their multifunctional actions. Hybrid

compounds and logical drug design approaches that target several pathogenic pathways implicated in AD are given particular attention. This study contributes to the ongoing efforts to discover more effective and disease-modifying therapies by providing useful insights into the design, synthesis, and biological assessment of new anti-Alzheimer drugs by reviewing the most recent experimental and computational investigations.

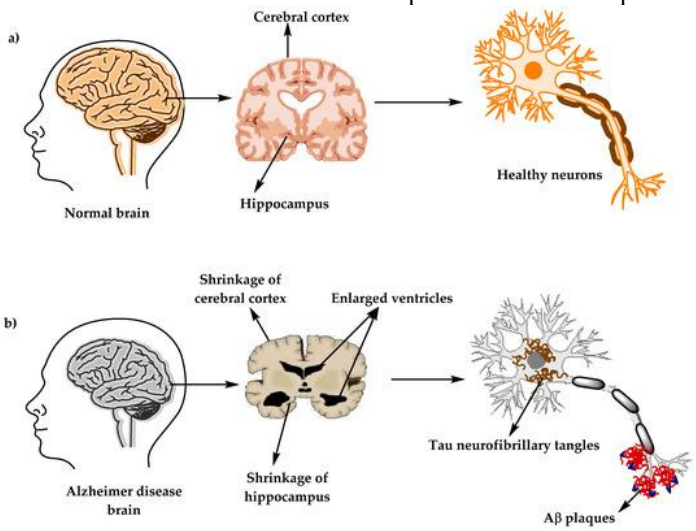



Figure (1) The physiological structure of the brain and neurons in (**a**) healthy brain and (**b**) Alzheimer's disease (AD) brain [13].

## 2. Alzheimer's Disease Therapeutic Targets:

Multiple biological processes interact intricately in Alzheimer's disease (AD), a multifactorial neurodegenerative illness. A comprehensive knowledge of the major molecular targets implicated in the course of illness is necessary for the development of successful treatment methods. The most common therapeutic targets in the treatment of AD are as follows:

### 2.1 Inhibition of AChE/BuChE:

The neurotransmitter acetylcholine, which is essential for memory and learning, is broken down by the enzyme acetylcholinesterase (AChE). Acetylcholine levels are markedly lowered in AD, which exacerbates cognitive deterioration. FDA-approved AChE inhibitors, such as galantamine, rivastigmine, and donepezil, function by raising acetylcholine levels in the synaptic cleft. Butyrylcholinesterase (BuChE) is another promising therapeutic target since its activity rises in later stages of the illness [14].

### 2.2 Aggregation of Beta-Amyloid:

One of the main characteristics of AD is the extracellular buildup of amyloid-beta (Aβ) plaques. The amyloid precursor protein (APP) is the source of Aβ, which can clump together to form harmful plaques that harm neurons. Treatments that target Aβ concentrate on increasing clearance pathways, reducing aggregation, or blocking its generation (e.g., BACE1 inhibitors) [15].

### 2.3 Aggregation of Tau Protein:

In AD, tau, a microtubule-associated protein, experiences hyperphosphorylation, which causes neurofibrillary tangles to develop inside neurons. These tangles cause neuronal death and hinder intracellular trafficking. Aggregation inhibitors and tau phosphorylation modulators, such the investigational substance RI-AG03, are examples of therapeutic strategies that target tau [16].

### 2.4 Neuro-inflammation and Oxidative Stress:

The pathophysiology of AD is significantly influenced by oxidative stress and persistent neuroinflammation. While chronic activation of microglia results in inflammation and neurodegeneration, excessive reactive oxygen species (ROS) harm neurons. To lessen these effects and maintain neuronal function, antioxidants and anti-inflammatory drugs have been studied [17].

### 2.5 Antagonism of NMDA Receptors:

Memory, learning, and excitatory neurotransmission are all influenced by N-methyl-D-aspartate (NMDA) receptors. Their overstimulation causes neuronal damage and calcium overload in AD. Clinically, memantine, an NMDA receptor antagonist, is used to reduce cognitive symptoms and avoid excitotoxicity [18].

### 2.6 Ligands Directed to Multiple Targets (MTDLs):

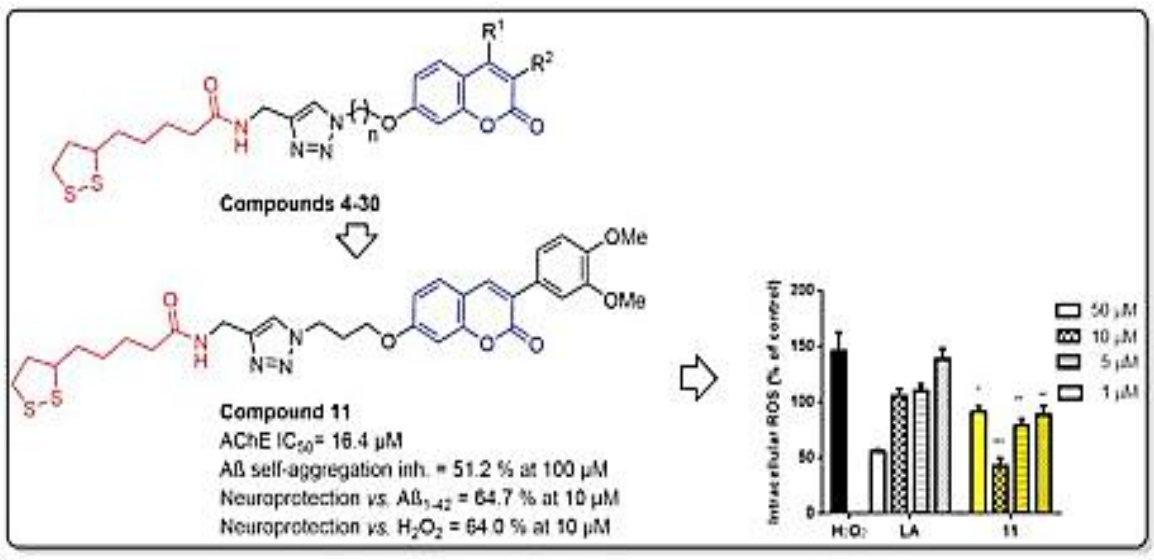
Multi-target-directed ligands (MTDLs) have become attractive therapeutic agents due to the complexity of AD. These substances are developed to concurrently alter several disease pathways, including metal chelation, antioxidant properties, anti-amyloid aggregation, and AChE inhibition. MTDLs provide better therapeutic potential and are a logical approach to addressing the complex character of AD [19].

## 3. Multi-Target-Directed Ligands (MTDLs):

Jalili-Baleh et al. (2018) developed and synthesized a variety of new multi-target-directed ligands by conjugating coumarin and lipoic acid scaffolds using alkyne-azide click chemistry, as shown in Scheme 1 [20]. The target compounds (4–30) were effectively synthesized using coupling processes, whereas the essential intermediates, 7-hydroxycoumarins, were generated using recognized synthetic techniques.

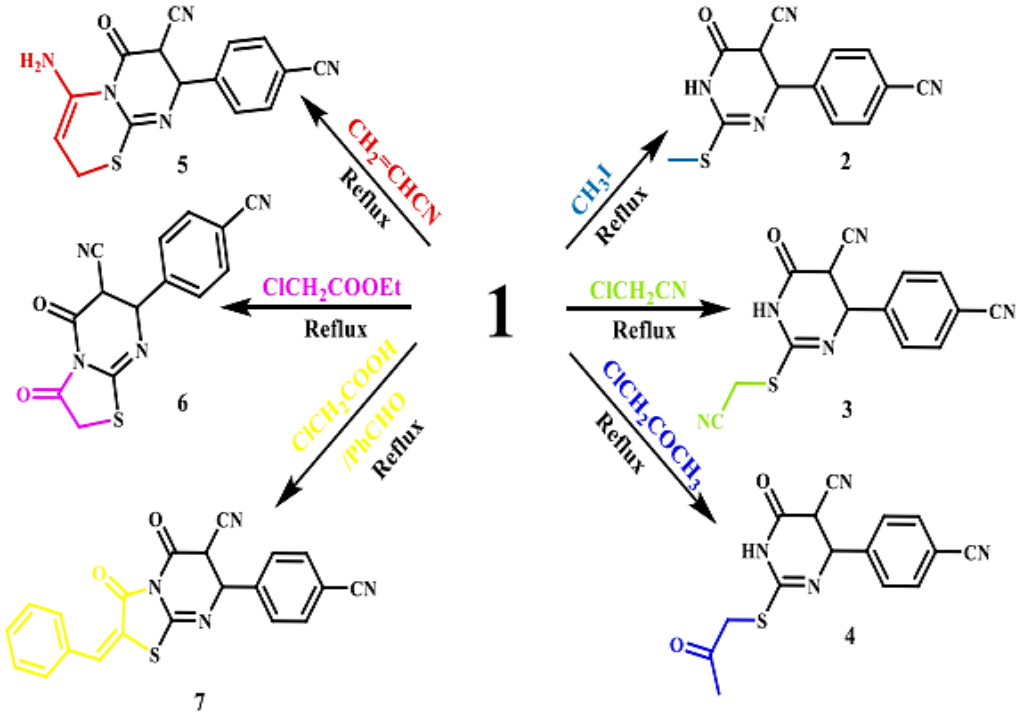
According to biological assessment, the majority of these compounds showed high antioxidant qualities and considerable neuroprotective efficacy against $H_2O_2$-induced oxidative stress in PC12 cells. Furthermore, compounds 11 and 30 had notable inhibitory effects in acetylcholinesterase (AChE) inhibition experiments. This study highlights the potential of coumarin-lipoic acid conjugates as multifunctional agents capable of simultaneously targeting several pathological features of Alzheimer's disease, including oxidative stress and cholinergic dysfunction. The work by Jalili-Baleh et al. exemplifies the promising multi-target-directed ligand

(MTDL) strategy to develop more effective disease-modifying therapies for AD.




Scheme (1): new multi-target-directed ligands by conjugating coumarin and lipoic acid [20]

In order to find possible anti-Alzheimer drugs, Shehab et al. investigated the synthesis and assessment of new pyrimidine derivatives utilizing manganese tetroxide nanoparticles ($Mn_3O_4$-NPs) as a catalyst. They produced 4-(4-cyanophenyl)-6-oxo-2 thioxohexahydropyrimidine-5-carbonitrile and its derivatives in moderate circumstances using an environmentally friendly and effective Biginelli multi-component reaction. The tumor necrosis factor-alpha (TNF-α) converting enzyme, a crucial mediator linked to neuroinflammation in Alzheimer's disease, was the target of molecular docking experiments conducted on these drugs. Promising binding affinities were found in the docking studies, confirming their ability to alter AD-related inflammatory pathways. Furthermore, the majority of produced compounds showed promising drug-like qualities according to pharmacokinetic profile, confirming their potential as therapeutic leads. This work is an example of an integrated strategy that combines pharmacokinetic evaluation, computational modeling, and nanoparticle-catalyzed synthesis to create multi-target-directed ligands (MTDLs) that can address the multifactorial nature of Alzheimer's disease, such as neuroinflammation and cholinergic dysfunction [21].



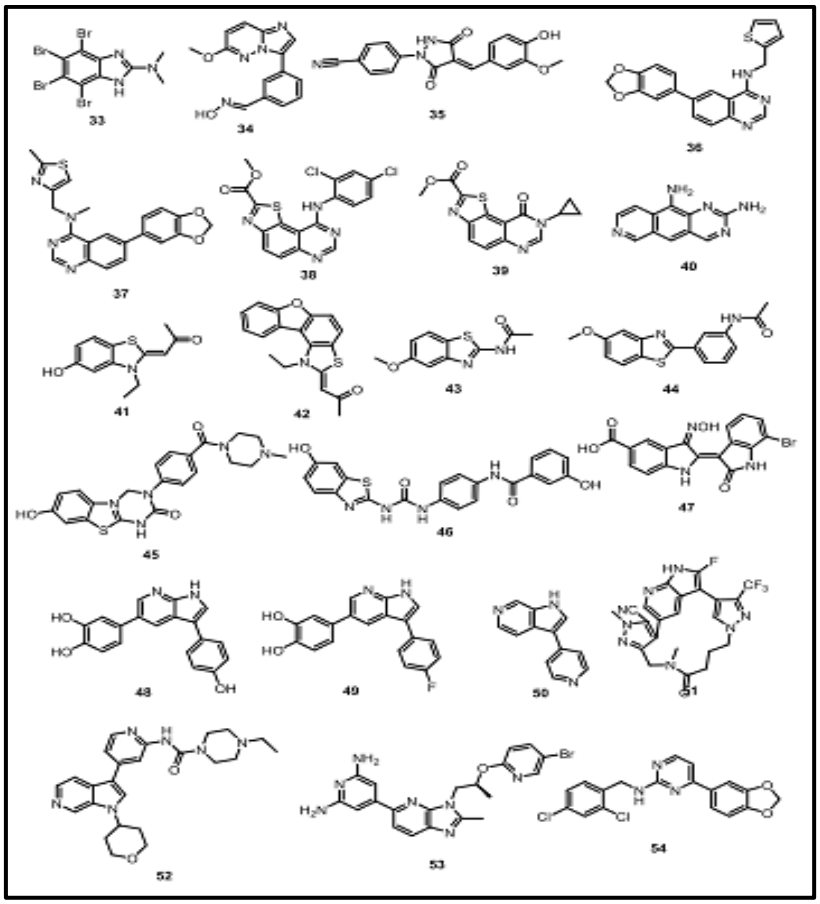

Scheme (2): the synthesis and assessment of new pyrimidine derivatives utilizing manganese tetroxide nanoparticles ($Mn_3O_4$-NPs) as a catalyst [21].

Numerous DYRK1A inhibitor classes have been created, each with a unique structure and strong action. Pagano et al. (2004) reported imidazole-based inhibitors after optimizing 4,5,6,7-tetrabromobenzimidazole derivatives and discovering compound 33, which had high selectivity across a kinase panel and an IC50 of 0.41 µM against DYRK1A [22]. In their evaluation of 3,6-disubstituted imidazo[1,2-b]pyridazines, Bendjeddou et al. (2017) found that compound 34 was a strong DYRK1A inhibitor (IC50 = 33 nM) that also had action against CDK1 and CLK2 [23]. When Koo et al. (2009) discovered compound 35 to be a DYRK1A inhibitor with an IC50 of 0.6 µM, pyrazolidine derivatives were born [24].

Quinazoline-based inhibitors have been thoroughly investigated; compound 36 was identified by Mott et al. (2009) as a CLK4 inhibitor (IC50 = 27 nM), while compound 37 was identified by Rosenthal et al. (2011) as a DYRK1A inhibitor (IC50 = 13 nM) [25, 26]. Among the thiazolo[5,4-f]quinazolines synthesized by Foucourt et al. (2014), compound 38 demonstrated the strongest DYRK1A inhibition to date (IC50 = 0.22 nM) [27]. Additional quinazoline derivatives with IC50 values of 91 nM and 28 nM, respectively, demonstrated DYRK1A activity and multi-kinase inhibition by Hédou et al. (2016) and Esvan et al. (2016) [28, 29]. Ogawa et al. (2010) were the first to use benzothiazole derivatives, and compound 41 (INDY) inhibited DYRK1A at 0.24 µM [30]. Compound 42 (BINDY), a strong selective inhibitor with an IC50 of 25.1 nM, was created by Masaki et al. (2015) [31]. Compound 43 (Rothweiler et al., 2016) and compound 44 (Stensen et al., 2021) are two further benzothiazole analogs that have demonstrated encouraging pharmacokinetic characteristics, including blood-brain barrier permeability [32, 33]. Additional benzothiazole-based selective inhibitors were described by Salah et al. (2018) and AlNajjar et al. (2022) [34, 35]. These compounds include compound 45 (IC50 = 95 nM) and compound 46, which also inhibits α-synuclein aggregation (IC50 = 7.8 µM).

Improved DYRK selectivity was demonstrated by indorubin derivatives created by Myrianthopoulos et al. (2013); compound 47 had an IC50 of 210 nM. Compound 48 (DANDY, IC50 = 3 nM, Gourdain et al. 2013), compound 49 (fluoro-DANDY, IC50 = 20.7 nM, Neumann et al. 2018), and compound 50 (Shaw et al. 2017) are examples of azaindole derivatives that have drawn attention for their potent inhibition of DYRK1A (IC50 = 1 nM) [36-39]. With IC50 values ranging from 3 to 6 nM, macrocyclic azaindole compounds like compound 51 (Powell et al. 2022) and compound 52 (Liu et al. 2020) considerably broadened this class [40, 41].

Weber et al. (2021) synthesized pyridine analogs and found compound 53 to be a strong and selective DYRK1A inhibitor (IC50 = 5 nM) [42]. Substituted pyrimidine analogs have also been thoroughly studied; Coombs et al. (2013) reported compound 54, which exhibited action against the DYRK and CLK kinases [43]. For DYRK1A, compound 53 showed an IC50 of 60 nM. Enantioselective pyrido[2,3-d]pyrimidines were reported by Anderson et al. (2013), with compound 55 obtaining IC50 values of 5 and 8 nM for DYRK1A and DYRK1B, respectively [44]. Among the 4,7-disubstituted pyrido[3,2-d]pyrimidines synthesized by Dehbi et al. (2014), compound 56 was a strong DYRK1A inhibitor (IC50 = 60 nM) that was also active against GSK3 and CD5 [45].



Scheme (3): Synthetic inhibitors of DYRK1A [46].

## 4. CONCLUSION

Alzheimer's disease is still a significant worldwide health concern because of its multifaceted, intricate character and the dearth of efficient disease-modifying therapies. Cholinergic deficiencies, beta-amyloid plaque buildup, tau protein hyperphosphorylation, oxidative stress, neuroinflammation, and dysregulated metal homeostasis are some of the pathogenic processes that contribute to the progressive neurodegeneration and cognitive loss linked to AD. The majority of the available treatments are symptomatic, highlighting the pressing need for new drugs that can target several disease pathways at once.

In this regard, the multi-target-directed ligand (MTDL) technique has become a viable method with the goal of creating artificial substances that can alter a number of AD pathogenic variables. A major step toward more potent treatments has been made with the creation of hybrid compounds that combine acetylcholinesterase inhibition, antioxidant qualities, anti-amyloid aggregation activity, metal chelation, and neuroprotective benefits. These multipurpose substances have the ability to alter the course of the disease in addition to reducing its symptoms

The viability and effectiveness of the MTDL technique are demonstrated by recent investigations that are discussed in this review, such as the synthesis of pyrimidine derivatives catalyzed by nanoparticles and coumarin-lipoic acid conjugates. Together with advantageous pharmacokinetic characteristics, these substances demonstrate potent biological activities such AChE inhibition, antioxidant capability, and neuroprotection. Moreover, kinase inhibitors that target DYRK1A and similar enzymes have demonstrated encouraging efficacy and selectivity, creating new opportunities to modify tau pathology and other molecular targets in AD.

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